molecular power. Posterior sclerosis could arise which might have no connection with the condition, but he thought that every one who had observed many cases would find that in after years the classic symptoms of tabes would develop.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, March 28, 1892.

The President, Dr. FRANCIS X. DERCUM, in the chair.

Dr. CHARLES W. BURR presented notes of

A CASE OF FRIEDREICH'S ATAXIA.—PATIENT EXHIBITED.

S. D., female, aged twenty-eight, single, born in Pennsylvania, admitted to the Philadelphia Home for nine months in May, 1891.

Family History.—Maternal grandmother living, aged eighty-two years, in good health. Paternal grandmother died of "old age." Maternal grandfather died of English cholera, paternal living, aged seventy-two, healthy. Mother in good health. Father killed by accident. A brother and sister living and in good health. One sister died in infancy. Careful questioning fails to reveal any nervous diseases in the family.

Personal History.—Patient is youngest child. Born at term. Labor easy. Instruments not used. Breast-fed. Never had convulsions. Menstruation began at fifteen years. No acute illness preceded present trouble.

Present Illness.—Patient's mother dates present illness from a fall which occurred when she was about eight years old. No serious injury resulted from the fall and the first symptom (staggering gait) did not appear until two years later. This difficulty in walking, with which later weakness was associated, gradually increased, until six years ago, since which time the patient has been chair-ridden. It is impossible to discover the order in which the other symptoms appeared, and the statement that staggering gait was the first may be erroneous. There have never been lancinating or other pains, crises, girdle sensation, or paræsthesias. Lateral curvature was noticed sixteen years ago. She has had no intercurrent disease. Some years ago chorea and later lateral spinal curvature were diagnosed.

Present State.—There is marked ataxia in arms and legs on movement, increased by closure of the eyes. On attempting to pick up a small object the hand waves hither and thither, passes beyond it, is brought back and hovers over it before grasping it. The same motion is, of course, shown on touching the nose or ear, and in putting the heel of one foot on the toes of the other. On attempting to sit with the back unsupported marked static ataxia appears; the trunk, arms, and head sway to and fro, and soon she sinks down into the chair. The movements are wide, somewhat jerky, and irregular. There is no tremor, either at rest or on voluntary motion.

There is some palsy in the legs. Talipes equino-varus is present. No palsy in arms. Knee-jerk and elbow-jerk are absent and are not re-enforcible. Plantar reflex is capricious. Chin-jerk is absent. Muscle-jerk in arms and legs is absent. Abdominal reflexes slightly present. Winking reflex present.

Sensation is normal in the arms. She localizes well and responds normally to touch, pain, heat, and cold. The same is true of the legs, except that on the toes localization is quite imperfect. The position of legs and arms passively moved with the eyes shut is well recognized. Speech is characteristic. Syllables are elicited and there is quite a great deal of hesitation. There is very slight wasting in the legs, no more than can be accounted for by disease and far too little to need involvement of the anterior horns for its explanation. Lateral spinal curvature with the convexity to the right is quite marked.

Dr. Charles S. Turnbull kindly examined her eyes, and informs me that the pupils are normal in size and react well to accommodation and light. There are no fundus lesions. No nystagmus is present; but on looking at an object near by and far to one side, there is at times a very slow and wide, irregular oscillation of the eyeball.

The abdominal and thoracic organs are normal. Menstruation normal. Urine normal. Mental condition excellent. During the nine months the patient has been under observation her condition has remained unchanged.

Cases of this disease are still sufficiently infrequent to merit publication. The main point of interest in the case is entire absence of neurological inheritance. The diagnosis of lateral curvature is also worthy of note, because it is probable that if orthopædic surgeons were on the lookout for this disease many more cases would be discovered. Within the past year there have been shown me two other cases in which the same error in diagnosis had been made.

DISCUSSION.

Dr. JAMES HENDRIE LLOYD.—I have for some time felt that I should like to see a clearer differentiation made between Friedreich's ataxia and the tabetic form of syringo-myelia, for I have a suspicion that some cases of so-called Friedreich's disease are simply cases of congenital failure of development and gliomatosis of the spinal cord. Bruhl, who has studied most of the reported cases, in his recent monograph on syringo-myelia states that it most closely simulates locomotor ataxia of all diseases.

The point that I want to make is, that where the cavity in the cord involves especially the posterior roots or posterior columns and does not extend forward and involve the anterior horns, there is established the tabetic form of syringo-myelia. The possibility of this condition has not been sufficiently recognized by writers on Friedreich's disease. Friedreich's disease is a disease of development, so is syringo-myelia. For some reasons there is a failure, in both these diseases, in the proper development of some portions of the spinal cord. In syringo-myelia it is not necessary that there should always be atrophy of the anterior horns. There is no more reason why we should always have a classical array of symptoms in this condition than that we should have it in any form of spinal tumor. Syringo-myelia is only a condition of gliomatosis, a proliferation of gliomatous tissue around the central canal. I think that if we knew the whole truth we should find that many cases of so-called Friedreich's disease were cases of the tabetic form of syringo-myelia.

In my wards at the Philadelphia Hospital I have an almost typical case of locomotor ataxia in a young person of twenty-four years. This disease has been slowly progressing since the age of fourteen years.

The patient has Argyle-Robertson pupil, atrophy of the optic nerves, fulgurent pains, scoliosis, and a tendency to slight atrophy of certain muscular groups. He has analgesia and areas of anæsthesia, but no thermo-anæsthesia. Whatever the morbid anatomy, his case suggests to my mind the possibility of some association between these two conditions, and of confusion in the reports of cases.

Many of the collected cases by Dr. Griffith have not been verified by post-mortem. A certain class of congenital or early hereditary cases of lack of development of the spinal cord are taken, and those that have a certain set of symptoms are placed in one class under the head of Friedreich's ataxia. I do not think that it is at all certain but that if we took a broader view of these cases, some of them would be found to be included under the head of a congenital lack of development of the cord closely analagous if not identical with syringo-myelia. I have no pathological foundation for asserting this positively, except the cases reported by other observers.

In Dr. Griffith's elaborate monograph 143 cases are tabulated, among which only 12 autopsies were made. In one of these a cerebral hemorrhage was found. Meningitis was observed in 10. The lateral pyramidal tracts were sclerosed throughout their whole length in 11 cases. The anterior pyramidal tracts were sclerosed in 6 cases. In 4 cases the central canal was filled with small round cells. "Supplementary canals" were observed in some cases. In one case such a canal was situated *behind* the main tube throughout nearly the whole lumbar region. In 3 cases our of these 12 autopsies true syringo-myelia was observed, as proved by this report, in which such canals, lined by columnar epithelium or replaced by gliomatous material are said to have been found.

In view of these post-mortem findings, as collected by Dr. Griffith himself, I think I am justified in asserting that many of the reported cases of Friedreich's ataxia do not represent a system disease *sui generis*, and that many of them are probably cases of syringo-myelia.

I wish it to be understood that I have not asserted that there is no such disease as a congenital or early-developed ataxia. I have simply raised the question of the differential diagnosis between Friedreich's ataxia and syringo-myelia. The question is not altogether a clinical, but partly an anatomical one.

The PRESIDENT.—The absence of arthropathies in Friedreich's disease is another interesting point.

Dr. J. P. CROZER GRIFFITH.—I agree fully with what Dr. Lloyd said about Friedreich's ataxia being a trouble connected with the development of the cord. There seems to be no question about that. Many cases have been reported in which it has appeared to be almost a congenital condition; since as soon as the child has made efforts at walking, difficulty has been observed. The occurrence of the disease in different members of the same family also points toward a congenital family weakness, particularly of the tissues of the posterior portion of the cord. The idea which was advanced by one of the French writers that this was not a condition of degeneration and connective-tissue growth, but a trouble with the neuroglia seems to have a great deal of truth in it, and this also would be in favor of the idea that the disease was due to a disturbance in the process of development. The idea has been advanced that the trouble is really an inherited tendency to vascular degeneration, and that as the blood supply to the posterior portion of the cord is not so great as that of the anterior portion the posterior half naturally suffers the most. In company with Dr. Burr I have been much interested in some observations relating to the condition of the cord in pernicious anæmia and in anæmia of other forms as well. These would indicate that possibly the blood supply has something to do with the development of posterior sclerosis; for the posterior portion seems to be more liable to suffer.

In typical cases of Friedreich's ataxia, as I consider Dr. Burr's case to be, there can be no difficulty in diagnosing the condition from syringo-myelia. In true Friedreich's disease a characteristic feature is that there is very little sensory disturbance. There is little or no pain. The girdle sensation is absent. The tactile sense is little affected. The muscle sense is not particularly involved. There are no arthropathies. The disease seems to be simply a progressive ataxia, spreading gradually from the lower to the upper portion of the body, and in some way the lesion does not extend above the medulla, but in some way speech is involved, and an "ataxic nystagmus," as Friedreich named it, is often present. This causes an oscillatory movement of the eyeball, which develops when the eye is fixed upon some object, particularly if to one side. The ordinary static nystagmus is rare.

There are some cases which differ from these typical cases. There have been a number such reported which I felt justified in including in a table of 145 cases which I collected and published some time ago. However, one can easily make mistakes, as I know well to my cost. A few years ago I saw, through the invitation of Dr. DaCosta, a case in which he had made the diagnosis of Friedreich's ataxia—a diagnosis in which I concurred, although the case presented some very anomalous symptoms. The autopsy showed that there was no lesion in the cord characteristic of the disease.

Friedreich's ataxia may teach us something in regard to the paths of conduction in the cord. This subject was brought up in a paper read by Dr. Inglis before the American Neurological Society last September. It is curious that in Friedreich's ataxia, with almost entire degeneration of the posterior columns, there should be so little disturbance of sensation, while in tabes dorsalis there should be so great disturbance. I do not understand why this should be so, unless possibly it is that sensation in the cord is conducted not in the posterior columns, but more in the border zone of Lissauer. Rütimeyer advances the theory, because in the two autopsies made by him this zone was affected, while in tabes dorsalis its involvement is one of the most constant lesions.

Another interesting matter which I do not entirely understand is that in the ataxia paraplegica of Gowers the symptoms should differ from those of Friedreich's ataxia, although the pathological appearances are almost identical.

In the paper to which I have referred I detailed the lesions found in the twelve autopsies which had been made up to that time. These lesions correspond in all important particulars. I think we are justified in saying that Friedreich's ataxia is a distinct combined systemic affection of the cord, just as much as tabes dorsalis is a systemic affection. We have no more right to deny the existence of this disease because some cases differ from the regular type, than we have to say that tabes dorsalis is not a distinct disease because cases have been observed and considered to be tabes dorsalis, and yet the autopsy has shown syphilitic meningitis.

It is true that in some of the autopsies some slight evidence of syringo-myelia existed. But it is also true that in some meningitis was present. In no case, however, do these lesions appear to have the slightest bearing upon the symptoms which had existed during life.

Both can only be regarded as accidental lesions, the principal lesion on which the disease depends being a systemic degeneration of certain portions of the cord, particularly of the posterior column and lateral tract.

Dr. D. D. STEWART by Dr. M. H. BOCHROCH presented for examination by the Society a

PROBABLE CASE OF SYRINGO-MYELIA.

Dr. CHARLES K. MILLS and Dr. J. B. DEAVER presented

SPECIMENS FROM A CASE OF TUMOR, PROBABLY GLIOMATOUS, OF THE CEREBELLUM, OBLONGATA, AND PONS, WITH HYDROCEPHALUS AND HYDRORACHIS.

Dr. AMELIA GILMORE read a paper on

INSANITY OF THE PUERPERIUM. (See page 408.)

DISCUSSION.

Dr. JAMES HENDRIE LLOYD.—One point in the etiology of insanity following labor which has always been of interest to me, is what is the proportion of cases in which there is a septic element. I think that sepsis is an important element in these cases, I am not prepared with figures, although on a former occasion I did go over this subject carefully. I think it was Clark who found, in studying a series of cases, that almost fifty per cent. exhibited some evidence of involvement of the womb or its surroundings—a true septic element. I do not mean to assert that this is so in all cases, but I believe that it is so in a certain proportion. In one case of insanity at the puerperium, in which I was called in consultation, there was well exhibited the conjoined effect of heredity, over-child bearing, anæmia, and sepsis. This woman had borne nine children in rapid succession, was overburdened with household cares, was anæmic, and had suffered from melancholic depression. After a troublesome labor she went into a condition of true sepsis, with elevation of temperature, a distended belly, metritis, cellulitis, and a typhoid tongue. This was her condition when I saw her four weeks after labor. She was sent to the Pennsylvania Hospital for the Insane, where she remained two years before she completely recovered. I think that the sepsis was a strong element in this case. I think, indeed, that many cases of confusional insanity or acute delirium support the view which I have before expressed that some of these cases are infectious in their nature.

The PRESIDENT.—Dr. Gilmore has alluded to the profound nervous exhaustion present in these cases. It seems to me that just for this reason they are particularly adapted to the rest cure. I am satisfied that the time devoted to treatment can be materially reduced, and, further, that cases otherwise hopeless can be cured by this means.

Dr. AMELIA GILMORE.—In regard to septicæmia, we find much less of it now than formerly. In many of the cases coming from the outside we do not know whether or not there has been a septic element. In the cases delivered in the hospital there is no septicæmia. I have seen no case which I could refer to sepsis.

In the treatment of these cases no regular method is adopted, but each case is studied by itself. The general treatment is that of rest, baths, special attention to diet, and special tonics and sedatives, as they are indicated.

Dr. WILLIAM EVANS read a paper entitled

SOME CLINICAL FEATURES OF SECONDARY CONTRACTURE IN BELL'S PALSY, AS ILLUSTRATED BY THREE CASES. (See p. 419.)

A Memoir of Dr. ANDREW J. PARKER was read by the President. A committee was appointed to prepare a minute on the death of Dr. Parker.

Adjourned.